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L7	L6 same l2	15	L7
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L3: Entry 2 of 9 File: PGPB Mar 28, 2002

PGPUB-DOCUMENT-NUMBER: 20020037274
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020037274 A1

TITLE: SINGLE AGENT METHOD FOR KILLING TUMOR AND TUMOR ASSOCIATED ENDOTHELIAL CELLS USING ADENOVIRAL MUTANTS

PUBLICATION-DATE: March 28, 2002

US-CL-CURRENT: 424/93.2; 435/320.1, 435/325, 435/375, 514/44

APPL-NO: 09/ 410462 [PALM]
DATE FILED: October 1, 1999
CONTINUED PROSECUTION APPLICATION: This is a publication of a continued prosecution application (CPA) filed under 37 CFR 1.53(d).

RELATED-US-APPL-DATA:
Application is a non-provisional-of-provisional application 60/105701, filed October 26, 1998,
Application is a non-provisional-of-provisional application 60/121300, filed February 23, 1999,

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L3: Entry 8 of 9 File: USPT Oct 14, 1997

DOCUMENT-IDENTIFIER: US 5677178 A
**** See image for Certificate of Correction ****
TITLE: *Cytopathic viruses for therapy and prophylaxis of neoplasia*

Brief Summary Text (18):
The invention provides novel recombinant adenovirus constructs which are replication defective in non-neoplastic cells but capable of expressing a replication phenotype in neoplastic cells lacking functional p53 and/or RB. The novel recombinant adenovirus constructs comprise a mutation, such as a deletion or point mutation, in the E1a and/or E1b gene regions, especially in the sequences encoding the E1b p55 protein and the CR1 and CR2 domains of the E1a p289R or p243R proteins. In some embodiments, a negative selectable gene, such as an HSV tk gene, is operably linked to an early region (e.g., E2, E1a, E1b) enhancer/promoter, a late region gene enhancer/promoter (e.g., major late promoter), or an early or late region promoter with a CMV enhancer, in a recombinant adenovirus construct also comprising an E1a or E1b mutation, so that *the negative selectable gene is preferentially transcribed in infected cells which* express a replication phenotype (i.e., neoplastic cells) and provides negative selection of such cells by administration of an effective dosage of a negative selection agent (e.g., gancyclovir, FIAU). A negative selectable gene may be inserted in place of an E1a and/or E1b structural sequence to concomitantly form an E1a.sup.(-) replication deficient mutant, E1b.sup.(-) replication deficient mutant, or E1a/E1b double mutant, respectively.

Detailed Description Text (22):
Typically, E1a-RB.sup.(-) replication deficient adenovirus constructs suitable for selective killing of RB(-) neoplastic cells comprise mutations (e.g., deletions, substitutions, frameshifts) which inactivate the ability of an E1a polypeptide to bind RB protein effectively. Such inactivating mutations typically occur in the E1a CR1 domain (amino acids 30-85 in Ad5: nucleotide positions 697-790) and/or the CR2 domain (*amino acids 120-139 in Ad5; nucleotide positions 920-967*), which are involved in binding the p105 RB protein and the p107 protein. Preferably, the CR3 domain (spanning amino acids 150-186) remains and is expressed as a truncated p289R polypeptide and is functional in transactivation of adenoviral early genes. FIG. 1 portrays schematically the domain structure of the E1a-289R polypeptide.

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L7: Entry 8 of 15 File: PGPB Oct 17, 2002

DOCUMENT-IDENTIFIER: US 20020150557 A1
TITLE: Selectively replicating viral vectors

Detail Description Paragraph (68):
[0098] Following infection of normal cells with 01/PEME, cellular p53 drives expression of E2F-Rb, antagonizing E2F function and inhibition of viral replication. In contrast, tumor cells with p53 inactivating mutations do not support expression of E2F-Rb from 01/PEME and thus allow replication of 01/PEME to proceed unhindered. Tumor cells with functional p53 also allow replication of 01/PEME, because their higher E2F activity facilitates transcription from E2F-dependent viral E1a and E2a promoters immediately after the virus enters the cells and prior to expression of sufficient quantities of E2F-Rb. Additionally, because inhibition by E2F-Rb is dependent upon competition with endogenous E2F activity, E2F-Rb would be less effective in inhibiting adenoviral early promoters when E2F activity is high as in tumor cells. In contrast, p53-dependent E2F-Rb expression would be effective in attenuating the virus propagation in normal cells because of both functional p53 and tightly regulated E2F pathways.

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L7: Entry 11 of 15 File: USPT Apr 8, 2003

DOCUMENT-IDENTIFIER: US 6544507 B2
TITLE: Anti-neoplastic viral agents

Brief Summary Text (10):
Further target specific defects are mutations of p16, cdk4, cyclin D or Rb (Bartek et al., 1997) in the retinoblastoma pathway which cause loss of G1/S control and essentially all tumours have these. The only significant exception is colon cancer, where mutations in the Rb pathway itself are rare. The net result of these defects is increased E2F activity, which means that tumours can be selectively targeted by viruses expressing toxic genes from E2F-regulated promoters. This has been demonstrated using an adenovirus expressing the HSV thymidine kinase gene from such a promoter (Parr et al., 1997); cells containing Rb-pathway mutations express tk and can be killed by ganciclovir. Such an approach relies on an increase in the activity of specific transcription factors in tumour cells.